

SIMULATION OF HEAD IMPACT LEADING TO TRAUMATIC BRAIN INJURY

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ABSTRACT

Traumatic brain injury, or TBI, is an unfortunate consequence of many civilian accident and military combat scenarios. Examples include head impact sustained in sports activities and automobile accidents as well as blast wave loading from detonated improvised explosive devices (IED). In the United States, over 5 million people live with disabilities associated with TBI (online report at www.cdc.gov/ncipc/tbi/TBI.htm). We present the results of a scoping study simulating the early-time wave interactions in the human head as a result of impact with a windshield in an automobile accident, a scenario leading to insipient conditions necessary for the onset of TBI. Our simulation results demonstrate that wave interactions within the head generate significant levels of stress at localized regions within the brain on an early time scale (~ 1 msec) prior to any overall motion of the head. The spatial distribution of these localized regions is consistent with the coup-contrecoup TBI mechanism observed in some patients that experience such impact events. In addition, smaller, localized regions with high stress occur in other parts of our brain model suggesting a mechanism for differential clinical outcome in TBI patients subjected to similar parameters of injury.

1. INTRODUCTION

Traumatic brain injury, or TBI, is associated with a loss of functional capability of the brain to perform cognitive and memory tasks, process information, and a variety of motor and coordination deficits. In many instances, the person involved in the event will not experience the full loss of brain function until days or weeks after the event has occurred. This suggests the existence of threshold levels and/or conditions of mechanical stress experienced by the brain that, if exceeded, lead to neural injury and evolving symptoms of TBI in the days or weeks following an accident.

To avoid a trial-and-error approach involving the large-scale use of laboratory animals to study various scenarios leading to TBI, we are developing numerical simulation models of the human head to study a spectrum

of impact and blast wave conditions that lead to the onset of TBI. In particular, we have established a collaborative effort between the MIND Imaging Center at the University of New Mexico and Sandia National Laboratories in order to create accurate models of the various tissues and geometries of the human head. With these models, we intend to conduct simulations of head impact that will permit us to establish a correlation between the incipient levels, rates, and duration of stress experienced by the brain and the onset of TBI.

In this paper, we present the results of a scoping study to simulate the early-time wave interactions occurring within the human head as a result of impact of an unrestrained person with the windshield of an automobile in a 34 mph head-on collision with a stationary barrier. Our three-dimensional head model was developed by importing a digitally processed, computed tomography (CT) scan of a healthy female head into the material definition package of the shock physics hydrocode CTH (Hertel, et al., 1993). Specifically, the CT scan was digitally processed to segment all soft tissue and bone into three distinct materials, skull, brain, and cerebral spinal fluid (CSF). Preliminary constitutive models were then formulated for the skull, brain, CSF, and windshield glass. The simulations were run on a parallel architecture computer employing 64 processors for each simulation run. The results of our simulations demonstrate the complexities of the wave interactions that occur between the skull, brain, and CSF as a result of the frontal impact with the glass windshield. These wave interactions result in the formation of localized regions within the brain that experience significant levels of pressure and deviatoric (shearing) stress. The details of these results will be discussed in the sections to follow.

A list of previous work focusing on the simulation of head impact leading to TBI includes that of Horgan, Gilchrist, and O'Donoghue, 2000, 2001, 2003, 2004, Nishimoto and Murakami, 2000, Suh, et al., 2005, Willinger, et al., 1999, 2003, Zhang, et al., 2001, Park and Yoon, 1997, Kleiven and von Holst, 2002, and Bandak, et al., 1995. Although there is some overlap in methodology of the current investigation with those works listed above, it appears that the present work is unique in employing

digitized CT scan data for head definition, sophisticated equation-of-state, strength, and fracture models to represent the biological materials, and an Eulerian, shock wave physics hydrocode with which to conduct our head impact simulations. Furthermore, the present work focuses on the early time (~ 1 msec) wave action occurring between the skull, brain, and CSF in order to investigate the connection between stress wave focusing and the ensuing brain injury due to localized damage.

2. METHODOLOGY

In order to conduct our simulations of head impact, the CTH hydrocode requires that material geometry and constitutive properties for all materials be provided as input. CTH employs a finite volume solution scheme that solves the conservation equations of mass, momentum and energy for each material on a spatial mesh fixed in space. The material geometry definition for the human head model was provided by digitally processing a CT scan data file such that the various biological materials comprising the head were segmented into skull, brain, and cerebral spinal fluid (CSF). Although the brain consists of a variety of tissue types (e.g., gray and white matter), delineated into compartments by the falx and tentorium membranes, it is our intention to start with a simplified representation in order to conduct scoping calculations. We expect to refine these models at a later date. Consequently, our brain model consists of homogeneous tissue possessing ventricular space containing the CSF (essentially water).

The CT scan data employed in this work consists of a stack of two-dimensional image planes perpendicular to the longitudinal axis of the head and body. The resolution of the CT scan was such that it provided high resolution within each axial plane but low resolution between planes. Consequently, the resulting material geometry for our head model occupies a three-dimensional mesh consisting of a stack of 51 two-dimensional planes possessing 512 cells per side leading to a total of roughly 13.4 million computational cells per calculation. This resulted in the mesh cell dimensions of $dx=dy=0.39$ mm and $dz=3.0$ mm. Within that mesh, we also inserted a glass plate, representing the windshield, positioned for frontal head impact. Figures 1 & 2 display the initial configuration of our impact simulations in both the sagittal plane and an axial plane positioned just above the eyes.

Assigning constitutive properties to the materials in our simulations requires that we provide an equation-of-state (EOS) to describe elastic pressure-volume response, a strength model defining the elastic & inelastic deviatoric (shear) response, and a fracture model representing the failure behavior for each material. The EOS representations for the skull, brain, and glass employed

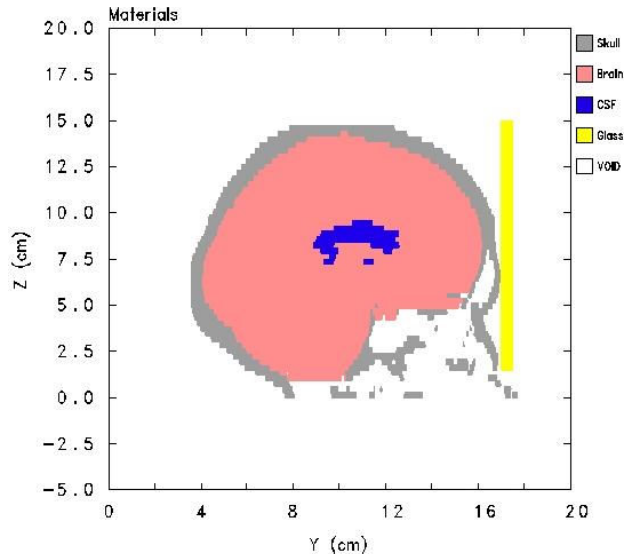


Fig. 1. Initial configuration of impact simulation; sagittal plane; glass located on right side.

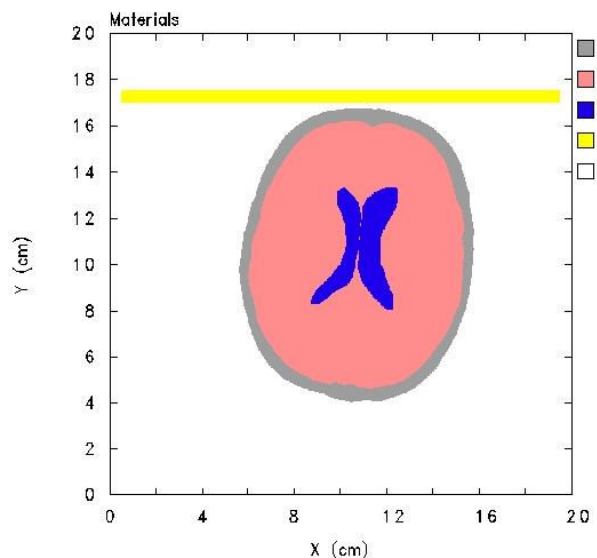


Fig. 2. Initial configuration of impact simulation; axial plane just above eyes; glass located at top.

the Mie-Gruneisen form (Hertel and Kerley, 1998) that defines the dependence of pressure and energy on mass density and temperature for materials subjected to shock wave loading. The Mie-Gruneisen EOS requires, as input, the specification of initial density ρ_o , specific heat C_V , Gruneisen parameter Γ_o , and the linear fit parameters, i.e., y-intercept (sound speed) C_s and slope S_1 , of the material's shock Hugoniot in particle velocity-shock velocity space. These quantities are presented in Table 1 for the skull (bone), brain, and glass. The CSF is represented by an EOS, in tabular form, that accurately represents the behavior of water in slightly compressed states in addition to capturing its vapor-liquid coexistence region of phase space.

Table 1. Mie-Gruneisen Equation of State Properties.

	ρ_o (g/cc)	C_s (km/s)	S_1	Γ_o	C_V (J/kg-K)
Skull	1.412	1.85	0.94	1.0	86.2
Brain	1.040	1.51	1.41	1.0	86.2
Glass	2.610	3.77	1.00	1.0	4808.5

The strength models define the deviatoric (shear) response (elastic and inelastic) for each material. In particular, we assume an elastic, perfectly plastic deviatoric response for the skull, brain, and glass. This assignment requires specification of shear modulus G and yield strength Y under conditions of uniaxial stress. The specific values of these parameters and the associated bulk modulus B for the skull, brain, and glass are listed in Table 2. The reader should note that since the CSF cannot support shear deformation due to its fluid nature, no strength model assignment is necessary for this material.

Table 2. Elastic & Inelastic Material Properties.

	B (GPa)	G (GPa)	Y (MPa)
Skull	4.82	3.32	95
Brain	2.37	0.48	20
Glass	37.1	36.4	200

In order to complete our definition of material behavior, we must assign fracture properties for each material. Since the skull (bone) and glass are considered brittle materials, we have employed the Johnson-Cook fracture model (Johnson, et al., 1985, 1989) to capture this behavior. This model defines a damage variable for each of its assigned materials. Damage ranges from 0 (no damage) to 1 (full damage) and is defined as the fraction of accumulated equivalent plastic strain up to a specific critical value. In the case of the skull (bone), the critical plastic strain at fracture has been assigned a value of 0.008; the critical strain for the glass was assigned a value of 0.047, based on reported fracture properties of tempered glass. As damage reaches its maximum value of 1, the fracture logic in CTH assumes that the material associated with the damage in question can no longer support tensile stresses and the effective strength of the material is significantly reduced to mimic failure. The failure properties for the brain tissue are based on a maximum tensile stress criterion. In particular, we assigned a tensile failure stress of 10 MPa for the brain tissue.

The initial conditions of the simulations assumed the head at rest and the glass plate striking the head slightly off-angle in a frontal impact with initial velocity of 15 m/sec (33.6 mph). Lagrangian tracer points were positioned at a variety of locations in the skull and brain to monitor local stress and motion histories. This allowed us to quickly identify any regions of elevated stress through the duration of the calculation.

The simulations were run on the Sandia Thunderbird computer system which consists of a 4480-node cluster totaling 8960 3.6GHz EM64T processors. Our particular simulations employed 32 nodes, running 2 processors each, in a parallel computation. The calculations were carried out to 1 msec of simulated time requiring 40 hours of CPU time to complete.

3. SIMULATION RESULTS

In our discussions with various medical researchers at the University of New Mexico, it became apparent to us that distinct cell damage mechanisms can occur, depending on whether the cells are subjected to isotropic stress (i.e., pressure) or shearing stress. Specifically, pressure imposes a volumetric or density change in the cell that can damage its internal structure. Shearing stress, on the other hand, tends to act as a tearing mechanism that damages the cell wall. Both of these damage mechanisms are at play in most incidents leading to TBI. As such, we present the results of our simulations by first analyzing the pressure distributions experienced in the head followed by the shear stress (deviatoric stress) results.

3.1 Pressure Results

After examination of the stress history data collected at the Lagrangian tracer positions placed throughout the head, we noticed that the pressure distributions suggest the classic coup-contrecoup insult to the head. That is, the frontal lobes of the brain experience significant compressive pressure as the shock wave propagates into the brain from the impact with the glass. This short-duration (0.2 msec) compressive wave propagates through the brain and reflects off of the skull at the rear of the head generating a tensile pressure wave which then propagates back into the brain. In particular, the frontal lobes sustain 25-30 bars of compressive pressure over a period of 0.1 msec early in the impact. The wave reflection off the rear portion of skull results in a tensile pressure of 3-4 bars in the posterior regions of the brain over a time period of approximately 0.1 msec. These results can be seen in Figures 3-6 which display the sagittal and axial views of the compressive pressure in the frontal brain lobes (Figures 3 & 4) and tensile pressure in the posterior regions of the brain (Figures 5 & 6).

In all of these figures, one notices the relatively large ventricular spaces within the central brain region. The ventricles contain the cerebral spinal fluid (CSF) that is distributed between various structures of the brain. The skull appears in all of these figures as the shell material surrounding the brain. At interfaces between the brain and skull as well as those between the brain and ventricles, our simulations predict the presence of small regions of elevated pressure, which appear at various times in the

calculation. This phenomenon is due to the shock impedance mismatch that exists between the different materials within the head, i.e., brain, skull, and CSF. This effect is more apparent in the plots of deviatoric (shearing) stress which is discussed in the next subsection.

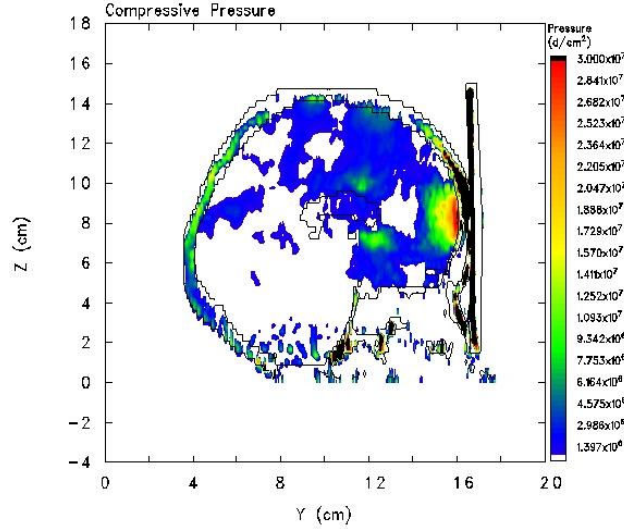


Fig. 3. Compressive pressure; sagittal view (glass at right); pressure scale: red: 30 bars, blue: 1 bar.

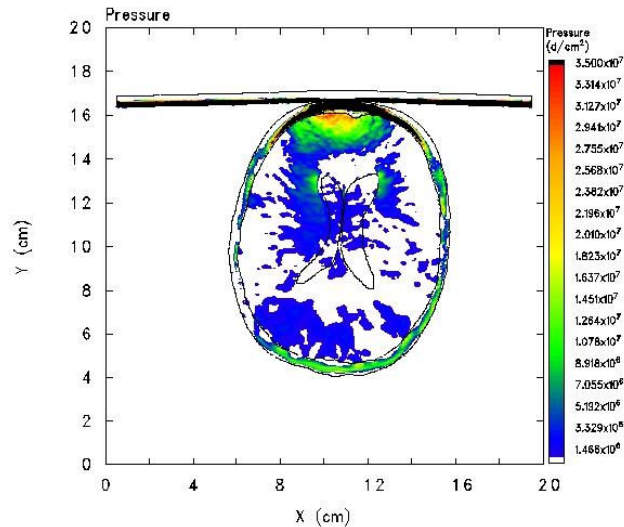


Fig. 4. Compressive pressure; axial view (glass at top); pressure scale: red: 35 bars, blue: 1 bar.

3.2 Deviatoric (Shearing) Stress Results

As with the pressure results, we examined the stress history data collected at the Lagrangian tracer positions in order to identify regions and times of significantly elevated deviatoric (shearing) stress. The results of this effort are displayed in Figures 7 & 8, which contain plots of the von Mises stress magnitude, which is always non-negative. The von Mises stress is directly related to the distortional, or shearing, strain that is experienced by any

material that can, in fact, support shear deformation. This type of stress results in a tearing action that, for biological materials, leads to cell membrane damage.

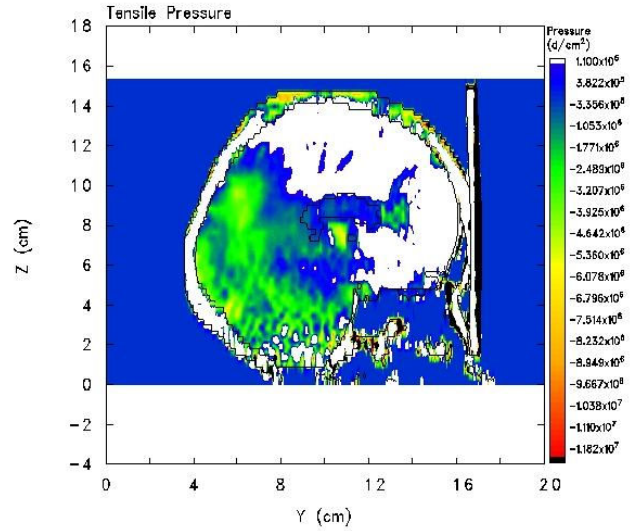


Fig. 5. Tensile pressure; sagittal view (glass at right); pressure scale: red: 12 bars tensile, blue: 1 bar compressive.

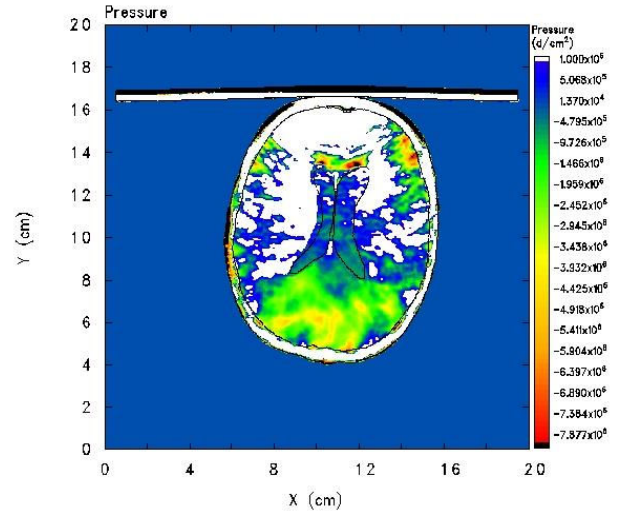


Fig. 6. Tensile pressure; axial view (glass at top); pressure scale: red: 8 bars tensile, blue: 1 bar compressive.

In particular, Figure 7 shows the von Mises stress distribution in the sagittal plane with the presence of a large frontal region of the brain experiencing stresses up to 30 bars. Figure 8 provides an axial view of the von Mises stress distribution for the same time (i.e., 0.4 msec) in the simulation as that depicted in Figure 7. Here, however, one can see the concentration of von Mises stress in the brain in proximity to the forward portions of the ventricles. It is interesting to note that the deviatoric stress is focused at regions of the brain adjacent to the ventricles since the cerebral spinal fluid, contained within the ventricles, cannot support shear deformation.

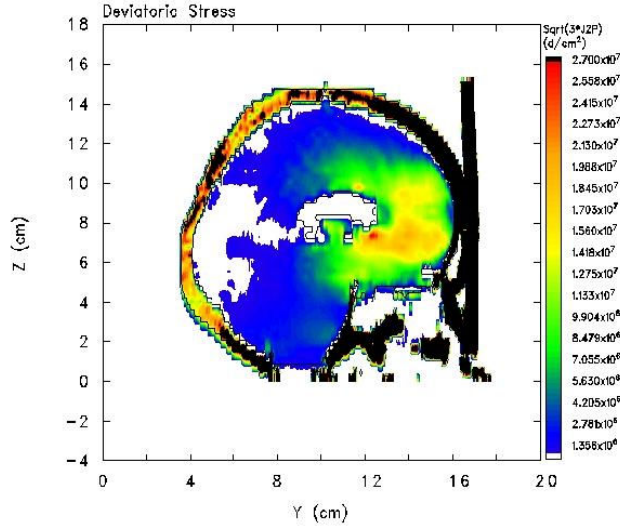


Fig. 7. Deviatoric stress; sagittal view (glass at right); von Mises stress scale: red: 27 bars, blue: 1 bar.

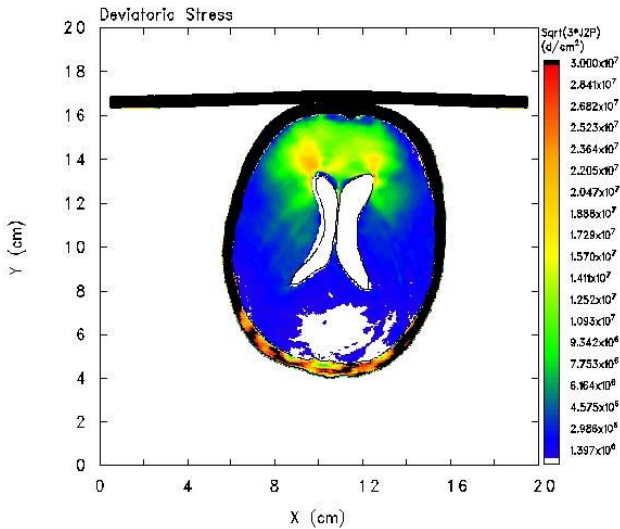


Fig. 8. Deviatoric stress; axial view (glass at top); von Mises stress scale: red: 30 bars, blue: 1 bar.

4. DISCUSSION OF RESULTS

As seen in section 3, our head impact simulations predict the classic coup-contrecoup insult when analyzing the pressure history results. This suggests that cell damage associated with this phenomenon is due to changes in cell volume. Furthermore, since the frontal regions of the brain experience compression whereas the posterior regions sustain both compression and tension, it is conceivable that different degrees of damage will result depending on the cell's tolerance of compressive versus tensile states of stress. Our simulations also predict the focusing of both compressive and tensile pressures at localized sites adjacent to the interfaces between the brain skull as well as those between the brain and ventricles.

Since, in reality, various membrane tissues are located at these interfaces, our results suggest that these membranes may experience damaging levels of stress as well.

The prediction of localized regions of elevated deviatoric stress around the ventricles suggests yet another mechanism and location for brain damage to occur. As mentioned earlier, deviatoric stresses tend to shear material. In this case, this type of stress would result in the tearing of cellular membranes. Since the brain is principally composed of neurons which conduct electrical impulses along their outer membrane, a tear in this membrane would be synonymous with the loss of electrical conductivity and hence, functionality.

CONCLUSIONS

We have conducted preliminary simulations of the early time (~1 msec) events associated with head impact that an unrestrained automobile occupant would experience with the windshield in a frontal collision at 15 m/sec (33.6 mph). We have chosen this scenario in order to study the insipient conditions leading to the onset of traumatic brain injury. Our results demonstrate the development of localized regions of elevated stress levels within the brain at the impact (coup) site, antipodal (contrecoup) site, and at the interfacial regions between the brain and skull as well as the brain and the ventricles. Furthermore, our simulations suggest the formation of at least two distinct mechanisms leading to brain damage. The first is associated with compressive and tensile pressures which cause cell volume changes leading to internal cellular damage. The second stress mechanism is related to the deviatoric or shearing stresses that develop at localized regions in the brain surrounding the ventricles. This state of stress tends to cause tearing of cellular membranes of brain neurons that lead to a loss of electrical impulse conductivity and cell functionality.

Perhaps one of the most important conclusions one can draw from these results is the fact that significant levels of stress are generated in the head during the impact event *before* any overall motion of the head occurs. Consequently, researchers simulating automobile accident scenarios need to also focus on the early time wave action occurring within the head in addition to the late time torsional and translational head motion that has been the traditionally focus of such studies.

Although we have demonstrated the ability to predict the focusing of stress within the brain due to an impact event, we cannot state what specific levels of stress will necessarily lead to TBI. In fact, this is precisely a future goal of our work - that is, the establishment of a quantitative correlation between the onset of TBI and the level, loading rate, and duration of pressure and deviatoric

stress within the brain. This would, in turn, lead to a TBI threshold criterion that could be used in the study of other insult scenarios to the human head. In particular, such a criterion could be used in the study of improvised explosive device (IED) blast effects to the head as well as providing a simulation tool to assess various armor strategies to mitigate the blast conditions leading to TBI.

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